Hormonal profiles, behavioral responses, and short-term growth performance after castration of pigs at three, six, nine, or twelve days of age[1,2]

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ABSTRACT: The objective of this study was to determine the effects of castration on short-term growth performance, hormone profiles, and behavior in pigs at 3, 6, 9, or 12 d of age. Ninety intact male pigs were assigned randomly to a treatment age by litter [3, 6, 9, or 12 d of age; n = 9 to 13 pigs per treatment (age) group]. Pigs within a single litter were then assigned to noncastrated (NC) or castrated (CAS) treatment groups according to BW. Pigs were nonsurgically fitted with jugular catheters, and blood samples were drawn immediately before castration (0 h) and at 0.5, 1, 1.5, 2, 24, and 48 h after castration. Body weights were obtained when pigs were catheterized and again at 24 and 48 h after castration. Serum samples were analyzed for cortisol, porcine corticosteroid-binding globulin, and dehydroepiandrosterone sulfate (DHEA-S). No differences were detected in initial BW of pigs, and there was no overall treatment effect on growth performance of pigs at 24 or 48 h posttreatment. A time × treatment interaction was detected ($P < 0.01$) for serum cortisol concentrations, such that cortisol was greater in CAS pigs than in NC pigs. No overall effect of age at castration was observed on cortisol concentrations. At 24 h after castration, serum cortisol concentrations returned to baseline in all treatment groups; however, at 48 h after castration, overall cortisol concentrations were elevated ($P < 0.01$) in the 6-, 9-, and 12-d-old pigs in both the CAS and NC groups compared with baseline concentrations. Total cortisol and porcine corticosteroid-binding globulin were used to calculate the free cortisol index (FCI). A time × treatment interaction was observed ($P < 0.01$) for FCI, such that FCI was greater in CAS males than in NC males. The FCI was also affected by age ($P < 0.01$). There was a time × treatment × age interaction ($P < 0.01$) for serum DHEA-S, such that DHEA-S concentrations decreased in CAS animals but increased in NC animals, and DHEA-S concentrations increased with age. During the first 2 h after castration, there was an overall age effect ($P = 0.01$) on the time that pigs spent standing, such that 3-d-old pigs stood more than 6-, 9-, or 12-d-old pigs. Treatment did not influence the time that pigs spent nursing, lying, standing, or sitting, although there was a trend ($P = 0.08$) for CAS pigs to be less active than NC pigs. These data indicate that castration is stressful regardless of age; however, the stress associated with handling seems to increase as pigs age.

Key words: behavior, castration, cortisol, dehydroepiandrosterone sulfate, growth, pig

 J. Anim. Sci. 2006. 84:1271–1278

INTRODUCTION

Pigs in US swine production systems are routinely castrated to eliminate boar taint in pork and to decrease aggressive behaviors and handling difficulties. The optimal age at which a pig should be castrated to minimize effects on performance and well being remains an area of debate in scientific and production arenas. Pigs castrated at several weeks of age, as opposed to several days of age, exhibit greater negative behavioral and physiological responses to castration (Wemelsfelder and Van Putten, 1985; McGlone and Hellman, 1988). Hence, castration before weaning is currently recommended (Widowski and Torrey, 2002). Nevertheless, in pigs <21 d of age, the optimal age to castrate is unclear.

The stress of castration can affect a number of factors, including behavior, endocrine and immune profiles, physiological responses, and growth performance. Previous studies reported acute behavioral changes in cas-

[1]The authors thank K. Holiman, P. Little, and M. Ellersick for technical assistance.

[2]Mention of trade names or proprietary products does not constitute a guarantee or warranty of the product by the USDA and does not imply its approval to the exclusion of other products that may also be suitable.

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Received September 14, 2005.
Accepted December 23, 2005.

trated pigs compared with intact male or female pigs (McGlone et al., 1993; Taylor et al., 2001; Hay et al., 2003), but no difference in behavioral responses of pigs with regard to age at castration has been determined (McGlone et al., 1993; Taylor et al., 2001). Performance results have been less consistent, and currently available reports are conflicting. McGlone et al. (1993) reported that pigs castrated at 14 d of age weighed more than pigs castrated at 1 d of age and had greater ADG when weaned at 28 d of age. In contrast, Kielly et al. (1999) found that growth of pigs castrated at 3 d of age was temporarily slowed compared with those castrated at 10 d of age, but no difference was found in weaning weight. Hay et al. (2003) also reported no effect of castration on growth performance in pigs castrated at 5 d of age vs. those left intact. Therefore, our objective was to determine effects of castration in 3-, 6-, 9-, or 12-d-old pigs using endocrine measurements, short-term growth performance, and behavior as indicators of stress and well being.

MATERIALS AND METHODS

Animal Procedures

The research protocol was reviewed and approved by the University of Missouri Animal Care and Use Committee. Ninety Landrace × Large White male pigs from primiparous sows were housed with their dams and female littermates in traditional 1.52-m × 2.13-m farrowing pens at the University of Missouri Swine Farm. Sows were fed twice daily according to NRC (1998) requirements for lactating sows. Twenty-four litters were assigned randomly to treatment age (3, 6, 9, or 12 d of age). Male pigs within a single litter (n = 2 to 9 male pigs per litter) were then assigned to castrated (**CAS**) or noncastrated (**NC**) treatment groups according to BW, for a total of 8 treatment groups: CAS at 3 d of age (n = 12); NC at 3 d of age (n = 11); CAS at 6 d of age (n = 12); NC at 6 d of age (n = 13); CAS at 9 d of age (n = 12); NC at 9 d of age (n = 12); CAS at 12 d of age (n = 9); NC at 12 d of age (n = 9).

All pigs were weighed and processed within 24 h of birth. Processing consisted of spraying the navels with Betadine solution (The Purdue Frederick Company, Stamford, CT) and administering 1 mL of iron dextran (VET LABS Limited, Inc., Lenexa, KS) i.m. in the neck. Pigs were not exposed to any of the other routine management procedures such as needle teeth clipping, ear notching, or tail docking. Individual animal identification was accomplished by numbering the pigs on the back with a large permanent marker. Three hours before treatment, a jugular catheter was inserted into male pigs (Carroll et al., 1999). At treatment initiation, pigs in the CAS group were held upside down, their scrotums were sprayed with Betadine solution, and they were castrated using a sterile scalpel blade. The incisions were sprayed with Betadine solution, after which they were returned to their pen. Pigs in the NC group were held upside down, sprayed with Betadine solution, and held for 45 to 60 s (approximately equal to the time taken for castration), and returned to their pen. A blood sample was drawn immediately before castration at 0 h and then at 0.5, 1, 1.5, 2, 24, and 48 h after castration. To evaluate the short-term growth performance responses after treatment, BW were obtained at 24 and 48 h after castration.

Serum Hormone Analyses

Serum was harvested from centrifuged blood and stored at –20°C until analyzed for total cortisol, porcine corticosteroid-binding globulin (**pCBG**), and dehydroepiandrosterone sulfate (**DHEA-S**). Serum concentration of cortisol was determined on duplicate samples in a single assay, which was previously validated in our laboratory (Daniel et al., 1999) using a Coat-a-Count kit according to the manufacturer's instructions (Diagnostic Products Co., Los Angeles, CA). The range of the assay was 0.5 to 50 ng/mL, and the sensitivity was 5 ng/mL. The intraassay CV was 9.1%. Serum concentration of DHEA-S was determined on duplicate samples in a single assay and was validated by our laboratory using coated tubes according to the manufacturer's instructions (DSL-3500, Diagnostic Systems Laboratories, Inc., Webster, TX). The range of the assay was 0 to 8,000 ng/mL. The assay sensitivity was 17 ng/mL, and the intraassay CV was 13.5%.

pCBG Analysis

Serum from blood samples collected at times 0 (pretreatment), 0.5, 1.5, and 24 h after treatment was analyzed for pCBG concentrations using an indirect ELISA method (Roberts et al., 2003). The intraassay CV was 9.5%.

Free Cortisol Index

Serum concentrations of pCBG were used along with total cortisol concentration to calculate the free cortisol index (**FCI**) at 0, 0.5, 1.5, and 24 h after castration. The FCI (measured in nmol/mg) was calculated by dividing the total cortisol (nmol/L) by the pCBG (mg/L; le Roux et al., 2003). To convert cortisol from traditional units (ng/mL) to System International units (nmol/L), which are necessary for calculating the FCI, cortisol values were multiplied by a conversion factor of 2.759 (Graham et al., 2002).

Behavior

Behavioral data were collected at 3-min scan sample intervals beginning immediately after castration and continuing for 2 h after castration. Behaviors were observed from time-lapse digital images (WJ-HD500A, Panasonic, Secaucus, NJ). To ensure full view of the pen, 2 cameras (WV-CP470, Panasonic) were mounted above each farrowing pen. Pigs were observed for the

Table 1. Description of behaviors analyzed

Behavior	Description
Active	Walking, running, playing, or fighting
Lying	Lying on belly with sternum in contact with floor or flat-out on side with shoulder in contact with floor
Lying – heat	Lying (as defined above) under the heat lamp or on the heat mat
Lying – total	Total time spent lying (lying plus lying – heat)
Sitting	Body weight supported by hindquarters and forelegs
Sitting – heat	Sitting (as defined above) under the heat lamp or on the heat mat
Sitting – total	Total time spent sitting (sitting plus sitting – heat)
Standing	Body weight supported by all 4 legs and pig remaining still
Standing – heat	Standing (as defined above) under the heat lamp or on the heat mat
Standing – total	Total time spent standing (standing plus standing – heat)
Nursing	Teat in the mouth coupled with active and rhythmic suckling motion

following mutually exclusive behaviors: active, lying, lying under the heat, sitting, sitting under the heat, standing, standing under the heat, and nursing (Table 1).

Statistical Analyses

Statistical analyses for growth performance, serum hormone, and pCBG concentrations as well as FCI were performed using Statview software (SAS Inst., Inc., Cary, NC); pig was the experimental unit. Growth performance data were analyzed by ANOVA for a completely randomized design with a 2 (CAS vs. NC) × 4 (age; d 3, 6, 9, or 12) factorial arrangement of treatments and all interactions. Cortisol, DHEA-S, pCBG, and FCI data were analyzed by ANOVA for repeated measures via a split-plot design with a 2 × 4 factorial arrangement of treatments in the main plot and time and all appropriate interactions in the subplot. Data are expressed as means (±SE).

Behavioral data were analyzed after arcsine-square root transformation for proportions using the GLM procedure of SAS; pen was the experimental unit. The data were analyzed as a split-plot; the main plot was age. The subplot contained the effects of treatment and age × treatment. Mean differences were ascertained using Fisher's LSD.

RESULTS

Growth Performance

The short-term growth performance during the first 24 and 48 h after treatment is presented in Table 2. Overall, no differences existed in initial BW of pigs among the treatment (age) groups. Additionally, regardless of age, no overall treatment effect was observed on growth performance of pigs at 24 or 48 h after treatment. Age affected ($P < 0.01$) BW, such that older pigs weighed more initially than younger pigs, but no treatment × age effect was detected.

Serum Cortisol

A time × treatment interaction was detected ($P < 0.001$) for serum cortisol concentrations, such that cortisol was greater in CAS males than in NC males (Figure 1) after treatment. A time × age interaction ($P < 0.001$) was associated with serum cortisol; overall serum cortisol was greater in the 9- and 12-d-old pigs than in the 3-d-old pigs ($P < 0.01$). Serum cortisol also tended ($P < 0.07$) to be greater in the 9- and 12-d-old pigs than in the 6-d-old pigs. However, an interaction of age and treatment was not observed for serum cortisol, as cortisol dramatically increased in all CAS pigs regardless of age by 0.5 h postcastration. At 24 h after castration, serum cortisol concentrations returned to baseline in both treatment groups regardless of age; however, at 48 h after castration, cortisol concentrations were increased ($P < 0.04$) compared with baseline concentrations in all age groups except the 3-d-old pigs.

Serum pCBG

Castration did not affect serum pCBG profiles (Table 3), but serum pCBG profiles varied over the sampling period ($P < 0.001$), as well as among the different age groups ($P < 0.002$). Mean serum concentrations of pCBG during the 24-h sampling period were 3.67 ± 0.22, 3.35 ± 0.18, 2.61 ± 0.16, and 2.52 ± 0.16 mg/L for the 3-, 6-, 9-, and 12-d-old pigs, respectively. Overall, serum pCBG concentrations decreased ($P < 0.05$) from 0 h (precastration; 3.69 ± 0.23 mg/L) to 24 h after castration (2.81 ± 0.17). At 0 h, serum pCBG concentrations were lower ($P < 0.05$) in 9- and 12-d-old pigs (3.08 ± 0.42 and 3.18 ± 0.40 mg/L, respectively) compared with 3-d-old pigs (4.55 ± 0.50 mg/L); however, at 0.5 h after castration, serum pCBG concentrations did not differ among any of the age groups. At 1.5 and 24 h after castration, serum pCBG concentrations were greater ($P < 0.05$) in 3- and 6-d-old pigs than in 9- and 12-d-old pigs.

FCI

A time × treatment interaction was detected ($P < 0.001$) for FCI, such that the FCI was greater in CAS pigs than in NC pigs at 0.5 ($P < 0.003$) and 1.5 h ($P = 0.001$) after castration (Figure 2). However, no time × treatment × age interaction was observed for FCI, although a tendency ($P = 0.07$) for a time × age interac-

Table 2. Acute growth performance during the first 48 h after treatment in 3-, 6-, 9-, and 12-d-old pigs[1]

	Beginning weight			Gain, 0 to 24 h			Gain, 24 to 48 h			Overall gain, 0 to 48 h		
Age[2]	NC	CAS	Main effect[3]	NC	CAS	Main effect[3]	NC	CAS	Main effect[3]	NC	CAS	Main effect[3]
3	1.85 ± 0.12	1.86 ± 0.11	>0.92	0.18 ± 0.03	0.16 ± 0.03	>0.62	0.14 ± 0.04	0.12 ± 0.02	>0.71	0.32 ± 0.07	0.28 ± 0.04	>0.65
6	2.38 ± 0.12	2.40 ± 0.15	>0.95	0.25 ± 0.02	0.26 ± 0.02	>0.78	0.14 ± 0.02	0.12 ± 0.03	>0.65	0.39 ± 0.04	0.38 ± 0.04	>0.88
9	2.76 ± 0.15	2.73 ± 0.19	>0.90	0.25 ± 0.02	0.25 ± 0.02	>0.81	0.08 ± 0.03	0.08 ± 0.04	>0.98	0.32 ± 0.04	0.33 ± 0.07	>0.98
12	3.75 ± 0.13	3.82 ± 0.25	>0.81	0.36 ± 0.06	0.32 ± 0.08	>0.71	0.14 ± 0.04	0.14 ± 0.04	>0.94	0.49 ± 0.04	0.46 ± 0.09	>0.75

[1]Values are means ± SE (kg) for noncastrated (NC) and castrated (CAS) pigs at 3 (n = 11 and 12 pigs per treatment, respectively), 6 (n = 13 and 12 pigs per treatment, respectively), 9 (n = 12 pigs per treatment), and 12 (n = 9 pigs per treatment) d of age.
[2]Age (d) of the pigs at the time of treatment.
[3]*P*-value for main effect of treatment (NC vs. CAS).

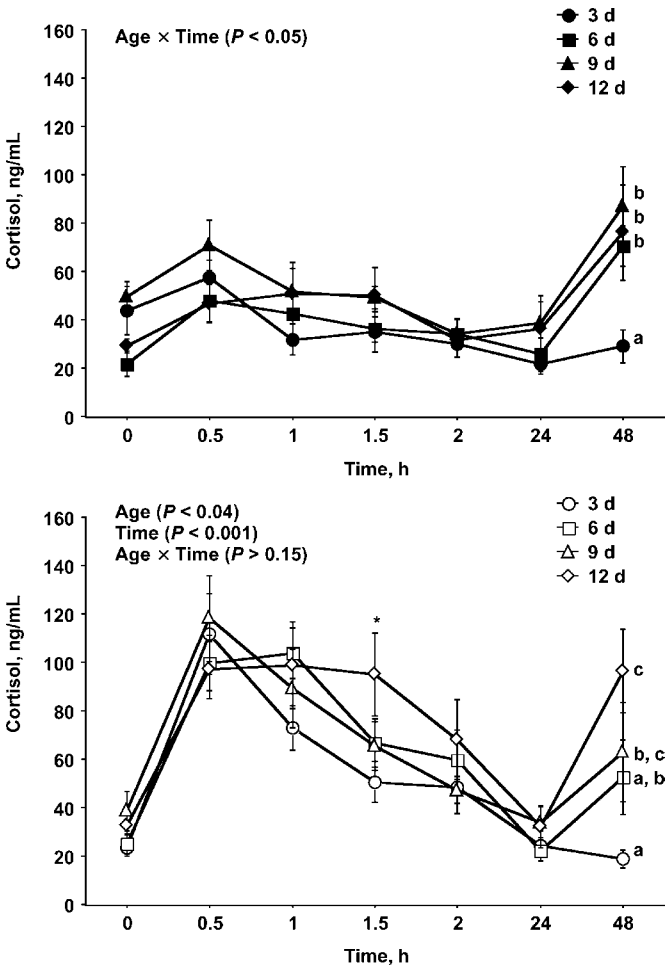

Figure 1. Serum concentrations of cortisol in noncastrated (top panel) and castrated (bottom panel) pigs at 3, 6, 9, and 12 d of age during the first 48 h after treatment. Treatments were applied immediately after the collection of the 0-h blood sample. In the noncastrated pigs (top panel), serum concentrations of cortisol at 48 h after treatment were greater in 6-, 9-, and 12-d-old pigs compared with 3-d-old pigs ($P < 0.04$). A similar cortisol profile was observed in the castrated pigs (bottom panel) at 48 h posttreatment a vs. b, a vs. c, and b vs. c: $P < 0.06$; 12 > 3, 6, and 9: $^*P < 0.09$).

tion was observed for FCI. At 0 h, 3-d-old pigs (25.1 ± 5.8 nmol/mg) had a lower ($P < 0.009$) FCI than did 9-d-old pigs (69.6 ± 13.7 nmol/mg), and 6-d-old pigs (22.7 ± 4.4 nmol/mg) had a lower ($P < 0.04$) FCI than both 9- and 12-d-old pigs (37.9 ± 4.9 nmol/mg). Although no differences were observed in the FCI among any of the age groups at 0.5 h after castration, at 1.5 h after castration, 3- (49.9 ± 8.4 nmol/mg), 6- (62.5 ± 10.3 nmol/mg), and 9-d-old pigs (79.6 ± 10.4 nmol/mg) all had a lower ($P < 0.05$) FCI than did 12-d-old pigs (135.2 ± 30.2 nmol/mg). Finally, at 24 h after castration, both 3- (20.4 ± 3.8 nmol/mg) and 6-d-old pigs (25.1 ± 4.5 nmol/mg) had a lower ($P < 0.05$) FCI than did 9- (69.6 ± 21.8 nmol/mg) and 12-d-old pigs (57.3 ± 14.2 nmol/mg). There were

Table 3. Serum porcine corticosteroid-binding globulin (pCBG) concentrations during the first 24 h after treatment in 3-, 6-, 9-, and 12-d-old pigs[1]

	0 h[2]		0.5 h[2]		1.5 h[2]		24 h[2]	
Age[3]	NC	CAS	NC	CAS	NC	CAS	NC	CAS
3	4.49 ± 0.71	4.60 ± 0.72	3.61 ± 0.57	2.92 ± 0.41	2.93 ± 0.45	2.75 ± 0.28	4.56 ± 0.95	3.57 ± 0.44
6	4.40 ± 0.70	3.23 ± 0.43	3.31 ± 0.40	3.44 ± 0.55	3.00 ± 0.37	3.38 ± 0.82	3.04 ± 0.25	2.98 ± 0.24
9	3.35 ± 0.78	2.8 ± 0.36	3.17 ± 0.64	3.13 ± 0.46	2.35 ± 0.23	2.01 ± 0.23	2.07 ± 0.20	1.94 ± 0.21
12	3.26 ± 0.68	3.10 ± 0.45	2.60 ± 0.41	2.96 ± 0.60	2.53 ± 0.38	1.66 ± 0.16	2.17 ± 0.19	1.89 ± 0.18

[1]Values are means ± SE (mg/L) for noncastrated (NC) and castrated (CAS) pigs at 3 (n = 11 and 12 pigs per treatment, respectively), 6 (n = 13 and 12 pigs per treatment, respectively), 9 (n = 12 pigs per treatment), and 12 (n = 9 pigs per treatment) d of age.
[2]Sample time (h) relative to treatment that occurred immediately after the 0-h sample.
[3]Age (d) of the pigs at the time of treatment.

no differences at 24 h in the FCI between 3- and 6-d-old pigs or 9- and 12-d-old pigs.

Serum DHEA-S

A time × treatment × age interaction was observed ($P < 0.01$) for serum DHEA-S (Figure 3). In the NC pigs, serum DHEA-S concentrations increased with age ($P < 0.001$) and remained relatively constant throughout the 48-h study (Figure 3; top panel). In CAS pigs, DHEA-S concentrations were greater in older animals than in younger ones before treatment; at 0 h, serum concentrations of DHEA-S were greater ($P < 0.04$) in the 9- and 12-d-old pigs than in the 3-d-old pigs and were greater ($P < 0.005$) in the 12-d-old pigs than in the 6- and 9-d-old pigs. Serum DHEA-S concentrations rapidly decreased ($P < 0.007$) during the first 30 min after castration in the 6-, 9-, and 12-d-old pigs (Figure 3; bottom panel). At 2 h after castration, serum concentrations of DHEA-S were still greater ($P < 0.02$) in the 12-d-old pigs than in the 3- and 6-d-old pigs. By 48 h after castra-

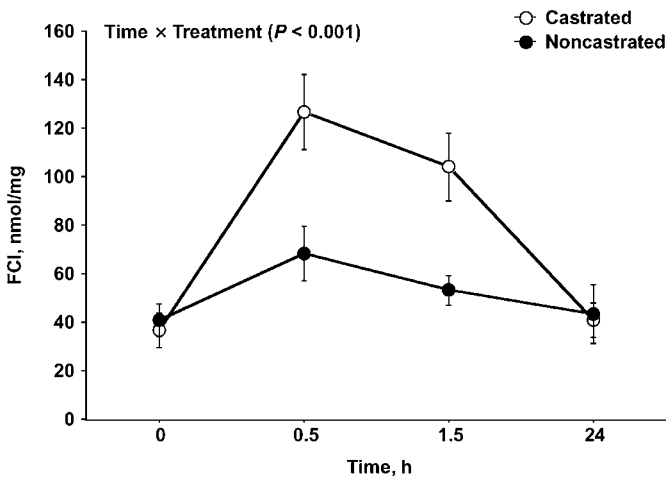

Figure 2. Free cortisol index (FCI) of noncastrated and castrated pigs during the first 24 h after treatment. Treatments were applied immediately after the collection of the 0-h blood sample. Free cortisol index (nmol/mg) was calculated by dividing serum cortisol concentrations (nmol/L) by porcine corticosteroid-binding globulin (mg/L) concentrations at each time point. There was an overall time × treatment effect ($P < 0.001$) on FCI, such that the FCI was greater in castrated pigs than in noncastrated pigs at 0.5 ($P < 0.003$) and 1.5 h ($P = 0.001$) after castration.

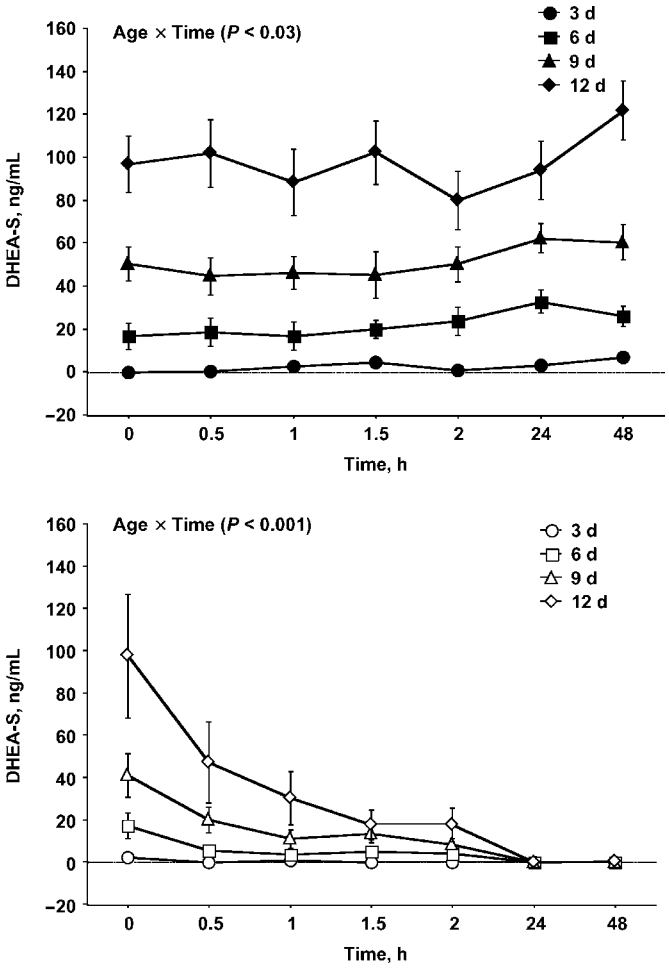

Figure 3. Blood serum concentrations of dehydroepiandrosterone sulfate (DHEA-S) in noncastrated pigs (top panel) and castrated pigs (bottom panel) pigs at 3, 6, 9, and 12 d of age during the first 48 h after treatment. Treatments were applied immediately after the collection of the 0-h blood sample.

tion, there was no effect of age on serum concentrations of DHEA-S.

Behavior

During the 2 h after castration, time pigs spent nursing, lying, standing, or sitting was similar in CAS and NC pigs, although a trend ($P = 0.08$) was noted for CAS pigs to be less active than NC pigs. Age affected ($P = 0.01$) time that pigs spent standing, such that 3-d-old pigs stood more than 6- ($P = 0.002$), 9- ($P = 0.012$), or 12-d-old pigs ($P = 0.01$). No age effect was apparent for the time pigs spent active, nursing, lying, or sitting.

DISCUSSION

In the current study, castration between 3 and 12 d of age did not influence the short-term growth performance (24 and 48 h after castration) of pigs. Whereas growth performance was evaluated during a relatively short period and on a limited number of pigs (45 castrated; 45 noncastrated), our results agree with those of Hay et al. (2003), who reported no difference in growth performance of 5-d-old castrated pigs compared with intact pigs weighed at 1, 2, 3, and 4 d after castration. Kielly et al. (1999) found that pigs castrated at 3 d of age weighed less than their weight-matched littermates 1 d after castration, but no difference was noted between pigs castrated at either 3 or 10 d of age compared with their weight-matched littermates at weaning. In contrast, McGlone et al. (1993) reported that pigs castrated at 1 d of age weighed less when weaned at 28 d than pigs castrated at 14 d of age. Castrated pigs in the current study tended to be less active than their NC littermates, which has been demonstrated in previous studies (Hay et al., 2003). Castrated pigs also have been found to miss more nursing bouts and suckle for decreased periods of time compared with noncastrated littermates (McGlone et al., 1993; Hay et al., 2003). These missed nursing opportunities have been suggested as the reason for decreased BW gain in pigs castrated at ≤3 d of age (McGlone and Hellman, 1988; Kielly et al., 1999; Hay et al., 2003). However, we observed no differences in nursing behavior among treatment groups, which would likely explain why we also did not observe a difference in acute growth performance. Our results agree with those of Taylor et al. (2001), who demonstrated no effect on nursing or growth performance because of castration in pigs at 3, 10, or 17 d of age.

Regarding the effect of age on the behavior of pigs after castration, conflicting data have been reported. Taylor et al. (2001) found that age of castration had no effect on behavior in pigs castrated at 3, 10, or 17 d of age, but that castrated pigs spent less time lying down and more time sitting or standing inactive than noncastrated pigs in the first 2 h after castration and less time lying in the subsequent 22 h after castration. Conversely, McGlone et al. (1993) found that castrated pigs spent more time lying down 6 h after castration than intact littermates, regardless of age, when castrated at 1 or 20 d of age. We found no difference in the time spent lying down in pigs castrated between 3 and 12 d of age; however, the time pigs spent standing differed according to age (but not treatment), as 3-d-old pigs spent the greatest time standing compared with older pigs. Previous studies reported no difference in standing behavior as a result of age (McGlone et al., 1993; Taylor et al., 2001).

Glucocorticoids are secreted in response to a stressor; therefore, increased concentrations of cortisol are generally considered indicative of stress (Sapolsky et al., 2000). The FCI, defined as the ratio of the serum concentration of total cortisol to CBG, is a good measure of serum free cortisol and is elevated in response to a stressor (le Roux et al., 2002). Because only free cortisol (that not bound to corticosteroid-binding globulin) is biologically active, the FCI has been found to be a more sensitive indication of the hypothalamic-pituitary-adrenal axis activation in some cases (le Roux et al., 2003). In the current study, elevated cortisol concentrations over time, as well as increased FCI in CAS pigs compared with NC pigs indicates that castration is a stressful experience. Nonetheless, we found that age at castration had no effect on serum cortisol, suggesting that castrating pigs between the ages of 3 and 12 d had no greater negative effects at one age vs. another and that castration is stressful regardless of age. Kattesh et al. (1996) collected a single blood sample at 3, 7, 10, 14, 17, 21, 28, 35, and 42 d of age from pigs castrated or not castrated at 7 or 14 d to determine cortisol concentrations and distribution in peripheral plasma. They found no differences in plasma cortisol distribution with respect to its bound or unbound form, and they concluded that because castration at 7 or 14 d did not change the normal physiological pattern of circulating cortisol, the pigs suffered no chronic stressful effects of the procedure. Because endocrine profiles fluctuate over time, collection of a single blood sample may not be indicative of the animal's response to stress. Serial blood samples, as performed in the current study, allowed for the compilation of more complete information regarding the stress response of pigs to castration (or simulated castration) compared with a single sample. In addition, because pigs in our study were fitted with nonsurgical indwelling jugular catheters for serial blood sampling, they were not repeatedly stuck with needles to obtain blood, which eliminated some of the stress associated with the blood collection process. The return of serum cortisol to baseline concentrations for all treatment groups at 24 h after castration indicates recovery from the stress of castration or simulated castration. However, the significant increase in cortisol concentration at 48 h for most of the treatment groups suggests that as pigs age, they have a greater stress response to being handled repeatedly compared with when they are younger.

It has been postulated that DHEA and DHEA-S function antagonistically with glucocorticoids (Herbert, 1998; Marx et al., 2003). Decreased DHEA and DHEA-S serum concentrations coupled with stable or increased serum glucocorticoid concentrations have been reported in both human and animal studies (Herbert, 1998; Straub et al., 2002; Marx et al., 2003). In the steroid hormone pathway, 17α-hydroxy-pregnenalone can be converted to either DHEA, which is readily converted to DHEA-S, or 17α-hydroxyprogesterone, which is converted to 11-deoxycortisol and subsequently to cortisol. Adrenal androgen production may be killed to achieve adequate serum cortisol concentrations necessary to respond to certain stressors (Herbert, 1998; Straub et al., 2002; Marx et al., 2003). The decrease in DHEA-S concentrations and subsequent increase in serum cortisol in the CAS animals may be necessary for castrated pigs to cope with the physical trauma of castration. In the NC pigs, although cortisol concentrations increased, the stress of simulated castration was evidently not sufficiently severe to elicit a change in the steroid pathway to increase glucocorticoid production at the expense of adrenal androgens, as seems to have been the case in the castrated animals. This hypothesis would explain the relative stability of DHEA-S in the NC pigs during the 2 h after castration, whereas DHEA-S in the CAS pigs decreased significantly. This also may serve as an explanation for the depletion of DHEA-S to undetectable concentrations at 24 and 48 h in CAS pigs.

Another possibility for the depletion of DHEA-S in the CAS pigs is that the majority of initial DHEA-S production occurs in the testes, which has been concluded in a number of studies (Neher and Wettstein, 1960; Huis in't Veld et al., 1964; Tan and Raeside, 1980). To determine whether the adrenals of pigs in this study secreted any significant amount of DHEA-S, we would need to have determined whether DHEA-S concentrations resumed a normal pattern of secretion once CAS pigs recovered from the stress of castration. We did not examine the adrenal glands or testes of the pigs in this study for DHEA-S concentrations; therefore, we cannot be certain of the source of DHEA-S. Nonetheless, based on earlier work (Neher and Wettstein, 1960; Huis in't Veld et al., 1964; Tan and Raeside, 1980) and the fact that DHEA-S decreased drastically in CAS pigs, the primary source of DHEA-S is most likely the testes. The adrenals of human infants produce minimal quantities of androgens, and it is not until the immediate prepubescent period that the production of DHEA-S and other androgens in the zona reticularis increases dramatically to where the adrenals in adult humans are responsible for 90% of DHEA-S production (Parker, 1999). Schwarzenberger et al. (1993) reported that DHEA-S secretion in male pigs peaked at 1 mo of age, decreased from 2 to 5 mo of age, and then increased again as pigs reached puberty. Sinclair et al. (2001) similarly found increased concentrations of plasma DHEA-S in pigs up to 2 wk of age, followed by a decrease, a plateau, and a gradual increase in the pubertal period as well. These findings corroborate the results of our study, in that serum DHEA-S concentrations increased initially in pigs from 3 to 12 d of age.

IMPLICATIONS

Minimizing stress to the young pig is essential for pig well being and optimal performance. Castration causes stress to the young pig, regardless of age; however, the present results indicate that as a pig ages, the stress caused by castration and/or handling increases. Therefore, when making management decisions with regard to procedures that require animal restraint, the potential for handling stress and how it is influenced by age should be considered.

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